

# Fluid and Electrolyte Disorders

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## NORMAL VOLUME HOMEOSTASIS

### Body Water

In the average adult, total body water is equal to 50% to 60% of body weight: 60% for men and 50% for women because of extra body fat, which is water free. In an average 70-kg man, total body water is 42 kg or 42 L, and for an average 70-kg woman, total body water is 35 kg or 35 L.

Approximately two thirds of total body water is located intracellularly and one third is located extracellularly. One fourth of the extracellular fluid volume (ECF) resides in the intravascular space. In a 70-kg man with 42 L of total body water, 28 L are located intracellularly, 14 L are located in the ECF, and 3.5 L are located in the extracellular intravascular compartment.

ECF volume is determined by the balance between sodium intake and excretion. Under normal circumstances, wide variations in salt intake lead to parallel changes in renal salt excretion, such that ECF volume and total body salt are maintained within narrow limits. This relative constancy of ECF volume is achieved by a series of afferent sensing systems, central integrative pathways, and renal and extrarenal effector mechanisms acting in concert to modulate sodium excretion by the kidney (Table 27-1).

The concentration of salt (i.e., sodium chloride [NaCl]) in the plasma is regulated by renal water handling. Plasma tonicity is maintained by sensing and effector mechanisms that are different from those that regulate volume, although the systems that regulate volume and plasma tonicity do work in concert. For example, if the baroreceptors of the body detect that the ECF volume is low, the kidney responds by retaining NaCl. This transiently leads to an increase in the tonicity of the ECF that stimulates release of arginine vasopressin (AVP), causing renal water retention and expansion of the ECF volume.

**TABLE 27-1** SENSORS AND EFFECTORS THAT DETERMINE OSMOREGULATION AND VOLUME REGULATION

FACTOR	OSMOREGULATION	VOLUME REGULATION
Sensors	Hypothalamic osmoreceptors	Low- and high-pressure baroreceptors
What is sensed	Plasma osmolality	Effective arterial blood volume
Effectors	Arginine vasopressin (AVP), thirst	Aldosterone, angiotensin II, sympathetic nerves
What is effected	Urine osmolality, thirst	Urine sodium (Na <sup>+</sup> ) excretion

## Osmolality and Tonicity

Osmolality is the number of particles per kilogram of solution. Plasma osmolality can be directly measured with the use of an osmometer or can be calculated by the following equation:

$$\text{Calculated osmolality} = (\text{Na}^+ \times 2) + \text{glucose}/18 + \text{BUN}/2.8$$

where Na<sup>+</sup> is the sodium ion concentration and BUN is the blood urea nitrogen level.

The osmolar gap is the difference between the measured and calculated osmolality and is normally less than 10 mOsm/L. A higher value indicates accumulation of an unmeasured substance such as ethanol, methanol, ethylene glycol, or acetone.

It is important to differentiate osmolality from tonicity. *Osmolality* refers to all particles in solution, and *tonicity* describes whether the particles are effective or ineffective osmoles. Effective osmoles such as Na<sup>+</sup>, glucose, or mannitol cannot penetrate cell membranes and can lead to changes in cell volume. Ineffective osmoles such as urea and alcohols pass freely into and out of cells and are unable to effect changes in cell volume. As an example, chronic kidney disease patients with BUN levels greater than 100 mg/dL have no cellular shifts of fluid due to the urea. The plasma osmolality is high, but plasma tonicity is normal.

## HYPONATREMIA

### Definition

Hyponatremia is one the most common electrolyte abnormalities encountered in clinical practice. Increasing age, medications, various disease states, and administration of hypotonic fluids are among the established risk factors for the disorder. Although hyponatremia is most commonly a marker of hypo-osmolality, three causes of hyponatremia are not associated with a hypo-osmolar state (Fig. 27-1). The first is pseudohyponatremia. This condition occurs in the setting of hyperglobulinemia or hypertriglyceridemia, in which plasma water relative to plasma solids is decreased in blood, decreasing the Na<sup>+</sup> concentration in a given volume of blood.

The second cause involves true hyponatremia with elevations in the concentration of an effective osmole. Clinical examples include hyperglycemia as seen in uncontrolled diabetes and infusion of mannitol for the treatment of cerebral edema. Increased plasma glucose concentration raises serum osmolality, which pulls water out of cells and dilutes the serum Na<sup>+</sup> concentration. For every 100-mg/dL rise in glucose or mannitol, the serum Na<sup>+</sup> level quickly falls by 1.6 mEq/L. The increased